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HEART DISEASE AMONG ELEMENTARY SCHOOL
CHILDREN.

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THE clinical picture of heart disease as seen in children who are well enough to attend school cannot fail to be of interest to the readers of this JOURNAL, differing as it does somewhat from the picture of cases seen in hospital. The latter picture forms the basis of all the text-book accounts of heart disease, which are a sort of composite photographs of innumerable more or less similar cases which have come under the observation of many different hospital physicians.

But the hospital cases nearly all have this in common, that the little patient was driven to the doctor by some discomfort or other, and in the very nature of the case was too poorly to be attending school at the time. Now that we have similar composite photographs derived from children well enough to attend school, and in a majority of instances totally unaware that they had anything wrong with their hearts, we shall be able to make some necessary adjustments in the final grand conception of heart disease in childhood.

The study of heart affections in school children is beset with difficulties. Take the case of diagnosis. A patient with heart disease in ordinary life seeks medical advice because of some discomfort—

breathlessness, or dropsy, or indigestion. When certain abnormal signs are detected in the heart there is usually no difficulty in connecting them with the discomfort by well-established lines of argument. The discomfort confirms one's impression of the true nature of the heart signs very much as a post-mortem examination does, though of course with less certainty.

The case is quite otherwise in school practice. In many cases there are no discomforts whatever; in others they are trivial or ambiguous. Therefore, diagnosis must depend entirely upon symptomatology, and the most experienced doctor must expect to have a fall sometimes.

We have to start with a clear idea of what is "normal" in symptomatology and then systematically challenge every deviation. Let us confine attention to endocardiac events. Exocardiac disease is very rarely seen or recognised in school work, and the occasional murmurs of exocardiac origin, such as the cardio-pulmonary and the murmurs generated by firm pressure of the stethoscope, are merely curiosities. The "normal" consists of (1) the placid "lub-dupp," heard, so to say, from the middle distance, neither just under the stethoscope nor feebly a long way off; (2) the absence of hurry or irregularity of rhythm; (3) the light touch and go of the apex beat under the extended hand, felt at or within, or at any rate not markedly outside, the left nipple line; this position of the apex beat should be verified by the test of percussion, for the apex beat is sometimes at the normal spot, when percussion reveals a margin of dulness beyond it.

These, then, in school work are the signs of a healthy, uninjured heart. Deviations from the normal take the well-known forms of murmurs, bruits, reduplications or accentuations of sounds; palpable thrills, heaving, pushing, slapping, or rapping modes of impulse; rhythm too quick or too slow, or irregular in time or force; heart sounds too faint or too loud; unusual position of apex beat, and increase or alteration of cardiac dulness.

When children are taken without selection at the routine inspections and inspectors are giving particular attention to the heart, deviations from the normal, trivial or otherwise, may be detected in as many as 40 per cent. of all cases, and I daresay even this large figure is not exhaustive. But no inspector troubles to make notes of all such signs on every occasion, though he may observe them. He will note all he thinks important, and some, but not all, of those he feels sure have no important meaning for the child's health. It will therefore be of interest to point out the number of cases which a

staff of inspectors experienced at the work, but not making a special research into minor heart symptoms, thought worthy of note. For four years we had a staff of inspectors, all of whom had had prolonged experience in general practice or in residential hospital posts before they came to us. Working over large groups of children numbering approximately 20,000 annually, the percentages of cases noted by all the inspectors together as having some heart affection were 6·2, 5·97, 7·17, 7·15 in the four successive years. This may be taken to represent the proportion of cases of heart affection which no one would deny to be worthy of note, out of the 40 per cent. of cases trivial or otherwise which we know to exist.

As to many of these abnormal heart signs or sounds, we have the vaguest notion how in detail they are produced. Who could give a satisfactory explanation of all cases of "thruppiness" or reduplication of the apex first sound? How is it that the heart "pushes" in one case, "slaps" in a second, and "raps" in a third? But there are other cases in which the signs and sounds so exactly resemble certain old acquaintances of the hospital ward which have been verified again and again at post-mortem examinations, that we feel no difficulty in looking on them as the same. On such evidence we attempt to divide the totality of abnormal signs into the *organic*, those which are due to some gross and permanent lesion, and the *functional*, those which arise in a substantially sound heart, or in one at least only temporarily defective.

That this effort at classification, especially in the circumstances of school inspection, is a precarious matter it would be vain to deny. It is not unlike the task of sifting the genuine cases of incipient pulmonary phthisis from the numerous cases of emaciated children with cough, catarrh, localised dull patches in the chest, chains of palpable neck glands, feverishness, and occasional night sweats, which nevertheless do not at any time give a positive von Pirquet reaction, and which by and by may clear up entirely, or in other cases remain the same for years.

A. ORGANIC CASES.—During the years 1912–13–14 Dr. Lila S. Greig, one of my colleagues on the school medical inspecting staff in Staffordshire, subjected the records of all cases of heart affection which had been noted at the routine inspections of 1908–9–10–11 to a careful examination. As we did not begin to revise old cases until 1912 the records scrutinised were mainly the original medical notes of a single examination.

Out of 70,138 routine children, boys and girls in equal proportions, and of the ages 5–6, 8–9, 12–13, 13–14, 674 had signs of

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organic valvular disease, 48 being considered to have congenital disease, and 626 acquired. In other words, the total amount of genuine organic valvular disease among Staffordshire school children would seem to be about 0·96 per cent.

Analysing a group of 676* apparently genuine organic cases we get the following proportions of murmurs :

	Cases.	Percentage.
Mitral regurgitant	562	83·1
Mitral stenotic	27	4·0
Mitral regurgitant and stenotic	49	7·3
Tricuspid	5	0·7
Pulmonary	26	3·9
Aortic	7	1·0
	676	100·0

This proportion of the various murmurs agrees in general with that found in hospital practice ; mitral regurgitant murmur is by far the most frequently found. Fortunately for the general accuracy of school statistics it is the least ambiguous of the murmurs. Taking all mitral valve murmurs together the preponderance is very large indeed.

One of the most surprising things in school medical work is the number of children with organic heart murmurs who seem to have no discomfort whatever. In many cases the announcement of the discovery is received with surprise and incredulity.

Revision of old cases is now done regularly, and we have case notes of 622 children with organic heart disease (valvular) which have been under observation for several years. More than half (55 per cent.) have never at any time during the period of observation, or so far as we know before, suffered from their ailment. A few of them have other ailments such as marked anæmia, phthisis, etc., not directly connected with their hearts, but no discomfort referable to the heart disease. Every variety of murmur is represented in this non-complaining group—congenital murmurs at pulmonary, mitral, and tricuspid valves, and mitral regurgitant, mitral stenotic, double mitral, aortic, among the post-natal cases.

Even this proportion of 55 per cent. free from complaints of any kind hardly does justice to the facts ; it leaves the picture still too gloomy. Every complaint mentioned by a child or put forward by

* This number consists of the above 674 organic cases minus a few but not all of the congenital cases, and plus a few cases of non-routine children specially presented because they had heart disease.

the parent or teacher which could by any stretch be ascribed to the heart is always counted in as if it were due to the heart alone, but in fact a large number of the complaints are trivial and ambiguous—"occasional malaise," "readily tired," and so forth. The complaining children out of this large group of 622 cases numbered 278, and the complaints were: Malaise alone, 145; cyanosis, 57; dyspnoea, 50; pain over heart, 30; headache, 20; fainting attacks, 17; œdema, 8; palpitation, 4; sickness, 3; sleeplessness, 2; clubbed fingers, 2; catarrhs, possibly due to venous stasis, 2; heart failure and loss of compensation, 2; epistaxis, 1; cough on exertion, 1; giddiness, 1. In a few cases the same child has two of these symptoms.

B. FUNCTIONAL CASES.—The group of functional cases, as already implied, is indefinitely more numerous than the organic. The signs and symptoms are most various; in fact, there is scarcely one of the deviations from the normal type of heart action referred to in an early paragraph of this paper which might not be safely regarded as functional if concurrent facts suggested it.

Dilatation.—Simple dilatation is meant without any obvious signs of organic disease. The fact of dilatation is indicated by the projection of the apex beat well beyond the left nipple line and by increase of the cardiac dull area in the same direction or to the right of the right edge of sternum. The absence of a massive heaving pulsation and of any organic murmur indicative of stenosis is commonly the ground on which we presume to say the case is not one of hypertrophy.

The area of cardiac dulness and the position of the apex beat are apt to vary from visit to visit in these cases. The pulse may be feeble or irregular, and there may be soft murmurs and bruits about the heart; but also there may be no such signs. The child is generally of a delicate, nervous type.

The number of cases of dilatation without obvious organic cause is not inconsiderable. Among 54,384 children, being a large portion of the routine children above mentioned, with the addition of a few hundreds of children "specially presented" by head teachers, there were 314 cases of simple dilatation (not hypertrophy), or 0.57 per cent.

The difficulty of feeling sure that a given case of dilatation has not behind it some true organic disease must always be allowed for. Out of 53 cases of supposed simple "functional" dilatation discovered among the routine children examined in 1911, which cases we were able to follow up at repeated visits until 1914, 10, or about 19 per cent., were in the latter year regarded as genuine organic

cases. Whether the organic features were present in 1911, but latent, or whether they had come on since, it was not always possible to say, but very likely in some cases they were present from the beginning.

The conceivable causes of simple dilatation are (1) a slight and passing toxæmia (such cases, if we could only recognise and define them, ought not, strictly speaking, to be called "functional"; they are disease lesions); (2) atony of neuro-muscular structures.

Murmurs and bruits heard over the præcordial area.—Humming, continuous bruits may be heard at the root of the neck or about the sterno-clavicular joints and are, of course, never taken to portend any organic disease. Equally "functional" are taken to be great numbers of gentle, sipping, sougling murmurs resembling interrupted bruits, systolic in time and heard at any of the valvular areas. They are particularly likely to be found at the pulmonary area. They are commonly thought to be present in the anæmic, but a surprising number of well-coloured children have them in typical form. One comes to note them and dismiss them from mind by an act of intuition, and it is not always easy to explain why one has so curtly dismissed them. If the murmur is soft, gentle, and little audible, if it is in the pulmonary area, if it is accompanied by similar soft murmurs at other valvular areas, if it is present at one inspection and not at the next, these circumstances one or all create a bias in favour of the "hæmic" or "functional" view. But, doubtless, mistakes must be sometimes made.

Accentuation and reduplication.—Accentuation of the second sound, mainly the pulmonary, is very common in childhood, as also is a reduplication of the first sound heard at the apex, converting "lub-dupp" into "thrub-dupp."

Unusual rate of heart-beat.—This may take the form of a somewhat regular alternation of a few rapid beats and a few slow ones (Mackenzie's "youthful type of irregularity"); or it may be the slipping of a beat, intermittency; or the intercalation of an unexpected beat between two normally spaced beats; or it may be the occurrence of an unusually forcible beat now and then.

Unusual method of impulse.—Instead of the normal light touch and go under the extended hand we may have a fuller and wider impulse, or a kind of slapping beat, or a diffused undulating beat short of the heavy massive heave of the hypertrophied heart; and it often has a hurried, excited character, suggesting over-action and "forcible feeble."

Although we cannot in many cases clearly imagine the mode of

production of many of these deviations from the normal which we class as functional, and to that extent our ideas of their ultimate causes must also be vague, yet one derives a strong impression from work among school children that in nearly all cases functional cardiac disorder is connected with that freakish instability of nervous system which is such a prominent factor in young life.

No paper on heart disease in elementary school children would be complete without a reference to a widespread condition (in its milder phases) commonly spoken of as "feeble circulation." It is really a local cyanosis of the extremities, cheeks, ear tips, etc. When strongly marked, the hands look as if they had been dipped to the wrists in some purple dye. Quite recently I came across a reference to a passage in Miss Ferriar's 'Marriage,' in which she speaks of "five awkward purple girls"; evidently they were cases of "feeble circulation," which has a marked tendency to run in families.

The condition has nothing whatever to do with feebleness of the central organs of the circulation; it is a vasomotor phenomenon and is apparently allied to Raynaud's disease; at any rate it would be possible to produce from among our Staffordshire school children a series of cases leading up from the lightest cases indistinguishable from "cold hands" (even when the day is not cold), by indefinite steps to a dangerous-looking local cyanosis and finally to true Raynaud's disease with gangrene, of which we have two or three cases at present on the rolls of our schools.

CONGENITAL TUBERCULOSIS—A SURVEY.

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(Continued from p. 332.)

SITZENFREY, in 1909, produced an elaborate monograph on "Congenital Tuberculosis with special consideration of Placental Tuberculosis." It included a review of the previous literature of the subject, to which I am much indebted. He himself investigated twenty-six placentas of tuberculous mothers, as well as the foetal membranes, the umbilical cords, and the newly born infants themselves, if they came to the post-mortem room. In seven of the twenty-six cases tuberculous changes or tubercle bacilli were discovered in the placenta or decidua vera, namely, as follows: In

three cases there were tuberculous lesions in the placenta; in one case there was a condition of endometritis decidualis tuberculosa, affecting the decidua vera and the decidua reflexa; in one case there was tuberculosis of the umbilical cord (the first recorded case with that lesion); in one case tubercle bacilli were detected in a vein of the decidua basalis and in a film of placental pulp; in the last of the seven positive cases tubercle bacilli were shown to be present in the placenta, in blood from the umbilical vein, and in the foetus itself, namely, in the capillary blood-vessels of the liver, in a retroperitoneal lymphatic gland and in a mesenteric lymphatic gland.

The localisation of the tubercle bacilli in the last-mentioned case in the foetal liver and lymphatic glands corresponded to observations of Kockel and Lungwitz and others on foetal tuberculosis in calves, and to the findings of Schmorl and Kockel in a human foetus, and F. Lehmann in a premature child that died one day after birth.

Sitzenfrey further noted that A. Gärtner had already suggested in 1893 that the embryonic cells and well-developed lymph-paths of the foetus allowed the easy passage of tubercle bacilli from the blood-vessels and organs to the lymphatic glands. This suggestion of Gärtner was confirmed by the above-mentioned observations of Kockel and Lungwitz (1894) on foetal tuberculosis in calves.

According to Sitzenfrey, the disease in the offspring seemed to some extent to be proportionate to the tuberculous changes in the placenta. In a case in which the placenta was full of miliary and larger tuberculous foci the child died four hours after birth. In a case with relatively few placental tubercles the child died, when three months old, from chronic pulmonary tuberculosis and bilateral tuberculous pleurisy. In another case in which only one tuberculous focus could be detected in the placenta the child had so far (first months of life) remained alive and well. The discovery of tuberculous changes in the placenta was therefore not necessarily a sign that the child would show signs of tuberculous infection during the first months of extra-uterine life.

In regard to the pathological changes in tuberculous placentas, Sitzenfrey laid special stress on the so-called "white infarcts" forming favourable ground for the development of tubercles.

Besides the already well-recognised way in which the foetus might become infected, that is to say, through its blood-stream from a placenta in which one or more tuberculous foci have "grown through" from the maternal to the foetal structures, Sitzenfrey explained two other methods by which the foetus could possibly

become tuberculous. He pointed out that chronic engorgement of the villous capillaries might lead to their rupture into the maternal intervillous spaces; and that lowering of blood-pressure in the villous tufts might permit the entrance of maternal blood, and allow the transference from the maternal to the foetal circulation of any tubercle bacilli which at the time happened to be free in the maternal blood-stream. His second suggested method was that tuberculous foci, localised in the decidua vera, might penetrate the amnion, infect the liquor amnii, and thus give rise to intra-uterine infection of the foetus with tubercle bacilli.

In regard to this last suggestion Sitzenfrey had been more or less anticipated by F. W. Andrewes, who, in January, 1903, when showing a specimen from the museum of Saint Bartholomew's Hospital, at a meeting of the Pathological Society of London, suggested that prenatal tuberculosis might in some cases possibly be caused by the foetus swallowing liquor amnii containing tubercle bacilli derived from a disintegrating lesion of the placenta. It had been already established that the entrance of amniotic fluid by the mouth might occur during intra-uterine life. The specimen exhibited by Andrewes consisted of the thoracic viscera of an infant that had died shortly after birth. The tracheal and bronchial glands were enlarged and caseous, and the lungs contained numerous miliary tubercles. The glandular lesions, according to Andrewes, must evidently have arisen long before birth.

Schlimpert (1910) gave the results of his examination of placentas from tuberculous women. Seven placentas were carefully and systematically examined. In five of them histological tuberculous changes were discovered and tubercle bacilli were likewise found present. In one of them tubercle bacilli were detected without any histological tuberculous changes. He pointed out that the presence of tuberculous changes in the placenta should be regarded as a reaction of the maternal tissues against the infection with tubercle bacilli.

Novak and Ranzel (1910) made a study of the placentas of tuberculous women, and by the help of the antiformin method they were able to demonstrate the presence of tubercle bacilli in the placenta in four out of six cases. The first of the four positive results was obtained with the placenta from a woman with advanced pulmonary and peritoneal tuberculosis. The child was born during the ninth month of pregnancy and appeared healthy. In the second positive case a remarkable feature was that the mother had signs of only slight tuberculosis of the apex of the (right) lung, without

sputum, and the puerperium ran a normal course, without fever. In the third positive case the pregnant woman suffered from very severe tuberculosis of the lungs, etc., and labour was induced at the fourth month. In the fourth case the mother suffered from miliary tuberculosis, dying very soon after the birth of an apparently healthy child.

A. Dietrich (1912) described the case of a servant-girl, aged 21 years, who died 3 days after childbirth, with disseminated miliary tuberculosis, apparently caused by the entry of tubercle bacilli into the blood-stream from tubercles in the wall of one of the right pulmonary veins. By the antiformin method tubercle bacilli were detected in the placenta. The child was removed from the mother directly after birth, and at first seemed to be healthy, but after nearly three months died with general tuberculosis. Dietrich regarded the child's case as one of *late* congenital tuberculosis in which the infection took place during labour (*intra-partum*), tearings or squeezings of the tuberculous placenta doubtless leading to the entry of tubercle bacilli into the foetal blood-stream, in a way similar to that suggested by A. Gärtner, H. Rietschel, A. Sitzenfrey, Pankow, and others.

A possibly similar case was described by G. Repaci, of Milan (in 1913), in which the child at birth was well developed and of normal appearance, but died on the thirty-ninth day of extra-uterine life with general miliary tuberculosis.

Perhaps the case described by Grulee and Harms (1915) might be explained in the same way. The child was apparently healthy at birth, but 15 hours after birth developed irregular pyrexia, which continued till its death (on the eleventh day) with generalised miliary tuberculosis, chiefly affecting the lymphatic glands near the liver, the liver itself, and the spleen. There was nothing pathological known in regard to the mother, excepting old tuberculous disease of the hip-joint, supposed to be healed. But cases like those of Repaci and of Grulee and Harms cannot be regarded as of *proved* congenital origin; nor of course can that of a child (recorded by H. Barbier, in 1915), who died at the age of $3\frac{1}{2}$ months, and was found to have, besides tuberculous broncho-pneumonia, unusually well-marked miliary tuberculosis of the liver; that organ was literally "stuffed" with miliary tubercles. Another doubtful example is that recorded by Bonnet (1898), in which the child of a gravely tuberculous woman died about three months after birth, with tuberculosis of lungs, intestine, mesenteric glands, liver, spleen, and kidneys.

H. Rollet (1913) recorded a remarkable case of intra-uterine miliary tuberculosis. The child died two days after birth (at full term) and was found to have generalised miliary tuberculosis, the miliary tubercles being especially numerous in the liver. There were caseous changes in many lymphatic glands in the thorax and abdomen, notably those at the hilus of the liver and in the mesenteric glands. The mother, aged 26 years, died from miliary tuberculosis about two weeks after childbirth, but no evidence of tuberculous infection of her uterus or of a fragment of retained placenta could be detected until tubercle bacilli were demonstrated in the latter, by the antiformin method.

Harbitz, of Christiania (1913), described the case of a child who died of pulmonary tuberculosis 25 days after birth. He regarded it as a case of congenital tuberculosis, in which the tuberculous infection of the foetus occurred during labour (*intra-partum*), in the way suggested by A. Gärtner, H. Rietschel, A. Sitzenfrey, etc. The mother, aged 26 years, had miliary tuberculosis, and died 28 days after childbirth. There was old tuberculous salpingitis. In the uterus, at the site of the placenta, there was a condition of tuberculous endometritis, so that during labour the foetus might easily have been infected by the entry into its blood-stream of tubercle bacilli from a ruptured tuberculous lesion in the placenta. The placenta itself was unfortunately not microscopically examined.

Péhu and Chalier (1908, 1914, and 1915) have analysed the whole subject of human congenital tuberculosis. They admit 51 published cases as conclusively genuine and as satisfying all critical demands. In all the cases that they admit the mothers have had grave exacerbations of tuberculosis and they regard it as plausible to assume that they all had acute generalisation of the disease. According to them and most other writers the father plays no part in congenital tuberculosis, all the cases being the result of a trans-placental "heredo-contagion," which latter, however, in comparison to tuberculosis acquired after birth, is excessively rare.

In the infected foetus or infant there are generally well-marked tuberculous lesions to be seen in the organs, but in some cases, in spite of no pathological-anatomical changes having been found, the foetal blood contained virulent tubercle bacilli. This latter condition is the "status bacillaris" of Honl, and is to be distinguished from the actual presence of tuberculous lesions in the foetus. The authors associate it with the occurrence of a terminal miliary tuberculosis in the mother, but surely the date and extent of the foetal infection have likewise much to do with it.

The authors say that tuberculosis of the placenta occurs in 45 per cent. of tuberculous mothers (according to F. Lehmann), but that microscopical evidence of the placental infection may require much searching. Apparently placental tuberculosis does not occur till the fourth month of pregnancy (*cf.* one of the cases recorded by Novak and Ranzel, in 1910).

In connection with this, Pankow's work (1910) is of special interest. In twenty pregnant tuberculous women artificial abortion was brought about in the third to fourth month of pregnancy, and from all these cases foetal pulp was injected into guinea-pigs, without giving rise to tuberculosis. In the case of two premature children of tuberculous mothers, at the seventh to eighth month of pregnancy, the guinea-pig inoculation test also proved negative in regard to the presence of congenital tuberculosis. Pankow expressed the opinion that many of the cases of congenital tuberculosis had arisen during labour, the tuberculous lesions in the placenta becoming ruptured by the labour "pains" (uterine contractions), and the transference of tubercle bacilli from the mother to the foetus being thereby facilitated.

In regard to the time at which tuberculosis of the placenta occurs one may note a case recorded by Dufour and Thiers (1913), in which the placenta appeared to be healthy though there was evidence of tuberculous infection in the foetus. A tuberculous young woman with hæmoptysis and meningitis, aged 19 years, died in the third to fourth month of pregnancy. The placenta appeared to be healthy, and examination for tubercle bacilli in it gave negative results; but the peritoneum of the foetus contained fluid, animal-inoculation with which gave rise to tuberculosis.

In regard to the possibility of "intra-partum" infection of the foetus or child with tubercle bacilli from the placenta, H. Rietschel's paper (1909) should be compared, and likewise the views of A. Gärtner (1893). With reference to these views, one might allude to the case of a child, aged 6 weeks, shown, in 1913, by M. Zarfl, at the Vienna Gesellschaft f. inn. Med. u. Kinderheilk. Von Pirquet's cuti-reaction for tuberculosis was positive on the seventeenth day of life, and increasing clinical signs of tuberculosis developed. Von Pirquet, who agreed with the diagnosis of congenital tuberculosis, said that in this case a positive tuberculin reaction had been obtained at an earlier age than in any other reported case. The case nevertheless can hardly be accepted as a proved one of congenital tuberculosis, for it might possibly have been acquired after birth.

Weller (1916) has reported two cases of miliary tuberculosis of the placenta, with latent tuberculosis in the mothers. In one of the mothers signs of ordinary pulmonary tuberculosis developed later on. The questions arising from the study of such cases are of considerable importance. In most cases of placental tuberculosis the mother has been suffering from advanced or miliary tuberculosis. Weller naturally refers to a case, somewhat similar to his cases, published by A. S. Warthin in 1913, under the heading, "Miliary Tuberculosis of the Placenta, with Incipient Pulmonary Tuberculosis of the Mother becoming Latent after the Birth of the Child." (*Cf.* also one of the cases recorded by Novak and Ranzel, in 1910.) It seems to me that we may well suppose that occasionally a mother with some form of chronic, and perhaps even latent, tuberculosis, may suffer from a temporary condition of tuberculous "bacillæmia" during pregnancy, without succumbing; for, probably, conditions of temporary tuberculous "bacillæmia" are not exceedingly rare in the subjects of chronic tuberculosis, apart from pregnancy.

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